Amendments to the Claims:

The following listing of claims will replace all prior versions and listings of claims in the application.

Listing of Claims:

1. (amended) A peptide having 2 to 10 amino acids, ~~or a derivative thereof,~~ which is able to restore wild type function of human p53 ~~for use in therapy~~.

Claims 2-26 (cancelled)

27. (new) The peptide of claim 1 having 3 to 7 amino acids.

28. (new) The peptide of claim 1 wherein the peptide incorporates the tri-peptide sequence WCT.

29. (new) The peptide of claim 28 wherein the peptide incorporates the pentapeptide sequence M-G/M/V-WCT.

30. (new) The peptide of claim 1 wherein the peptide has been modified at the C and/or N terminus to include a signaling or targeting moiety.

31. (new) The peptide of claim 30 wherein the signaling or targeting moiety is selected from the group comprising folate and the HIV Tat translocation sequence.

32. (new) A method of treating cancer comprising administering to a patient in need thereof a peptide having 2 to 10 amino acids which is able to restore wild type function of human p53.

33. (new) The method of claim 32 wherein the peptide has 3 to 7 amino acids.

34. (new) The method of claim 32 wherein the peptide incorporates the tri-peptide sequence WCT.

35. (new) The method of claim 34 wherein the peptide incorporates the pentapeptide sequence M-G/M/V-WCT.

36. (new) The method of claim 32, wherein the peptide has been modified at the C and/or N terminus to include a signaling or targeting moiety.

37. (new) The method of claim 36 wherein the signaling or targeting moiety is selected from the group comprising folate and the HIV Tat translocation sequence.

38. (new) A method of screening a library of molecules for the ability of members of the library to restore or modify the function of a target protein in an intra-cellular environment, the method comprising introducing the library into host cells having a reporter system that allows for the identification of those cells in which the function of the target protein has been restored or modified.

39. (new) The method of claim 38 wherein the target protein is a nucleic acid binding protein.

40. (new) The method of claim 39 wherein the nucleic acid binding protein is p53.

41. (new) The method of claim 38 wherein the reporter system comprises a reporter gene which is operably linked to a sequence of nucleotides that provides a binding site for the target protein or for a protein that associates with, or is a substrate for, the target protein.

42. (new) The method of claim 41 wherein the reporter gene is operably linked to a p21 or Bax promoter.

43. (new) The method of claim 41 wherein the protein product of the reporter gene includes a secretion signal peptide.

44. (new) The method of claim 41 wherein the protein product of the reporter gene includes a transmembrane domain.

45. (new) The method of claim 41 wherein the host cells have been transfected with the reporter gene.

46. (new) The method of claim 38 wherein the molecular library is a peptide library.

47. (new) The method of claim 46 wherein the peptides have 2-8 amino acids.

48. (new) The method of claim 46 wherein the library is introduced into the host cells in the form of nucleic acid constructs which encode the peptide library.

49. (new) The method of claim 46 wherein each member of the peptide library has the sequence M-G/M/V-$(X)_n$, wherein n is an integer from 3 to 18, M is methionine, G is glycine, V is valine and each X, which may be the same or different, is any genetically coded amino acid.

50. (new) The method of claim 38 wherein the molecular library has at least 500 different members.

51. (new) The method of claim 38 wherein the host cells are eukaryotic cells.

52. (new) A pharmaceutical composition comprising a compound, identified by a method according to claim 38, which is able to restore wild-type function to a mutant protein.

53. (new) A method of treatment, wherein the condition is selected from cancer, cystic fibrosis, sickle cell anemia, phenylketonuria, multiple carboxylase deficiency, methylpurine DNA glycosylasedeficiency (MPG), ataxia and chemotherapy resistance due to mutations in the gene coding for methylguanine-DNA methyl transferase (MGMT), comprising administering to a patient in need thereof the pharmaceutical composition of claim 52.